HAEMATOLOGICAL, HISTOPATHOLOGICAL AND GROWTH CONDITIONS OF AFRICAN CATFISH (*Clarias gariepinus*) JUVENILES EXPOSED TO SUB-LETHAL CONCENTRATIONS OF CHROMIUM

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ABSTRACT

This study examined the effects of sub-lethal chromium (Cr) exposure on haematological parameters, pathology and growth performance of juvenile *Clarias gariepinus*. Based on the LC50 value of 29.6 mmg/L, four hundred juveniles were exposed to Chromium concentrations of 0.00, 0.50, 1.00, 1.50 and 2.00 mg/L for 28 days, while maintaining water quality within optimal ranges. Haematological assessments revealed significant, dose-dependent reductions in packed cell volume, haemoglobin and white blood cells. At 2.00 mg/L, packed cell volume decreased from 36.00% in the control group to 17.50% (51.39% reduction), while haemoglobin decreased from 15.00 g/dL to 5.00 g/dL, representing a 66.67% decrease. White blood cell counts dropped by 66.67%, from 13.50×10^3/µL to 4.50×10^3/µL. Growth performance was adversely affected, with mean weight gain reducing from 15 g in the control group to 9 g at 1.50 mg/L chromium concentration, and specific growth rate decreasing from 2.73% to 1.84%. The percentage weight gain dropped from 115.09% in the control to 67.30% at the highest Chromium concentration. Histopathological examination revealed concentration-dependent damage to the gill, liver and muscle tissues. Observed lesions included epithelial lifting, lamellar fusion in the gills, hepatocellular vacuolation in the liver, and myofiber disorganization in muscle tissues, with the severity of these lesions increasing with higher Chromium concentrations. Thus, sub-lethal chromium exposure induced significant haematological disruptions, tissue damage, and reduced growth in *Clarias gariepinus*, with potential implications for ecosystem health and human consumption safety.

Keywords: Chromium, Haematology, Histopathology, *Clarias gariepinus*

INTRODUCTION

Aquatic ecosystems which are critical for sustaining biodiversity, face increasing contamination from both natural and anthropogenic sources, such as industrial effluents, domestic waste, and agrochemicals. Chromium, which is a heavy metal of concern, is released from anthropogenic sources such as tanneries, textile industries, electroplating, metal finishing and pigment manufacturing, all of which contribute significantly to aquatic pollution (Cervantes *et al.*, 2001; Shanker *et al*., 2005; Sharma *et al.,* 2020). Other pollutants of concern include cadmium, mercury, lead, industrial solvents, and emerging contaminants such as microplastics, nanoparticles, and pharmaceuticals (Hughes *et al*., 2013; Larsson, 2014; Malaj *et al*., 2014).

As pollutants accumulate in aquatic environments, fish which is a bioindicator, bioaccumulate toxic substances, leading to physiological and pathological disruptions (Farkas *et al.,* 2002; Authman *et al.,* 2015). Histopathological assessment of organs such as gills, liver and kidneys, are sensitive indicators of toxicant exposure and its adverse effects on fish health (Cengiz, 2006; Shah and Parveen, 2022). Likewise, haematological assessments provide early detection of physiological stress caused by environmental pollutants, making them valuable tools in aquatic toxicology (Samprath *et al.,* 1993; Ahmed *et al.,* 2022). These methodologies help in the monitoring of contamination levels and their impacts on fish, especially in the face of increasing pollution (Parek and Tank, 2015; Akbary *et al*., 2018).

The African catfish (*Clarias gariepinus*) is widely cultured in Africa, particularly Nigeria. The species was selected for this study due to its economic significance and resilience to environmental stressors, including metal contamination. Although the species is now predominantly obtained from aquaculture systems in Nigeria, it can still be exposed to chromium through contaminated water used in culture ponds, feed ingredients, and effluents that may seep into aquaculture facilities from nearby industrial sources. This makes *Clarias gariepinus* a suitable model for assessing the chronic impacts of chromium pollution. Investigating the haematological and histopathological responses of *Clarias gariepinus* to chromium provides insights into the broader effects of pollution on aquatic ecosystems, with important implications for food safety and public health.

MATERIALS AND METHODS

Experimental Setup

The study was conducted at the Department of Aquaculture and Fisheries Management, University of Ibadan, Nigeria. The study adhered to the bioassay method for long-term toxicity testing in aquatic environments as described by FAO (1986). Analytical-grade potassium dichromate ($K_2Cr_2O_7$) was used to prepare sub-lethal chromium concentrations in dechlorinated water.

LC50 Determination

The median lethal concentration (LC50) of chromium for *Clarias gariepinus* juveniles was determined through a 96-hour static renewal bioassay, following protocols established by Finney (1971) and APHA (2017). Fish were exposed to varying chromium concentrations, with mortality recorded at 24, 48, 72 and 96 hour intervals. Probit analysis revealed the LC50 to be 29.63 mg/L.

Experimental Fish and Acclimatization

A total of 400 juvenile *Clarias gariepinus* (mean weight: 13±0.05 g) were sourced from a reputable farm in Ibadan, Nigeria. The fish were acclimated for 14 days in circular plastic aquaria, with regular water changes, and fed commercial pelleted feed containing 45% crude protein (Adom Ltd, Ibadan). Feeding was discontinued 24 hours prior to the start of the experiment (Oyelese and Faturoti, 1995).

Experimental Design

The study utilized a completely randomized design with five treatment groups (0.0 mg/L, 0.5 mg/L, 1.0 mg/L, 1.5 mg/L and 2.0 mg/L),

each replicated three times in fifteen plastic tanks and 20 fish per replicate. Water quality parameters (temperature, dissolved oxygen, pH and conductivity) were measured using a mercury-in-glass thermometer, HI 3810 Dissolved Oxygen Test Kit, ATC pH meter (009), and a conductivity meter, respectively (Boyd, 1979; Eaton and Franson, 2005; APHA, 2017).

Haematological and Histopathological Analysis

Blood samples were collected from selected fish at 7, 14 and 28 days after exposure. The parameters measured were done, following the methodologies outlined by Sarma (1990) and Thrall *et al.* (2012).

Fish samples were collected on the 1st and 28th day, from each treatment group to extract liver, gill and muscle tissues. These samples were fixed in 10% neutral buffered formalin for histological analysis, following the method described by Akpokodje and Mpama (2005). The organs were then processed at the Department of Veterinary Pathology, University of Ibadan.

Histological preparations were as follows:

1. *Fixation:* Tissues were fixed in 10% neutral buffered formalin to preserve cellular structure.
2. *Dehydration*: Gradual removal of water using an automated tissue processor with ethanol concentrations (70%, 80%, 90%, 95%, and 100%) for 1 hour each.
3. *Clearing*: Ethanol was replaced with xylene (2 hours) to prepare tissues for embedding.
4. *Infiltration*: Tissues were infiltrated with molten paraffin wax (2 hours per wax change) to provide support for sectioning.
5. *Embedding:* Processed tissues were oriented and embedded in paraffin wax, then allowed to solidify.
6. *Sectioning:* Tissues were cut into 4µm thick sections using a microtome, mounted on frosted glass slides, and dewaxed at 40°C for 30 minutes.
7. *Staining:* Sections were stained with Haematoxylin and Eosin, then mounted with DPX.

Growth Parameters

Growth parameters were calculated using the following formulae:

- Mean weight gain (g) = $final\ weight - initial\ weight$
- Relative weight gain (%) = $\frac{final\ weight - initial\ weight}{initial\ weight} \times 100$
- Specific growth rate (SGR%) = $\frac{\log final\ weight - \log initial\ weight}{Days} \times 100$
- Feed conversion ratio (FCR) = $\frac{dry\ feed\ consumed}{weight\ gain}$
- Survival rate (SR%) = $\frac{initial\ fish - final\ dead\ fish}{initial\ fish} \times 100$

(Ricker, 1979; Tucker and Hargreaves, 2009).

Statistical Analysis

The data collected for haematology and growth parameters were subjected to one-way analyses of variance and significantly

different means were separated using Least Significant Difference (LSD) and compared using Duncan's Multiple Range Test (DMRT) (Steele and Torrie, 1980).

RESULTS

Haematological Analysis

Exposure to various concentrations of chromium significantly affected the haematological parameters of *Clarias gariepinus* juveniles. The control treatment consistently showed the highest haematological values, which decreased significantly with increasing Chromium concentrations.

After 7 days, the control group exhibited the highest PCV (33.00±1.41%), while the 2 mg/L Chromium group had the lowest (22.50±0.70%). Haemoglobin was highest in the control (11.50±0.70 g/dL) and lowest in the 2 mg/L group (8.50±0.70 g/dL). Red Blood Cell counts showed a similar trend, with the control recording 9.50±0.70 × 10^6/µL compared to 4.50 × 10^6/µL in the 2 mg/L group. White Blood Cell counts followed this pattern, with the control group showing 11.50 ± 0.70 × 10^3/µL and the 2 mg/L group being 8.00 ± 4.24 × 10^3/µL. Mean Corpuscular Haemoglobin (MCH) and percentages of lymphocytes, neutrophils, and monocytes also showed significant reductions with increased Chromium concentrations (Table 1).

By 14 days, similar trends were observed. The PCV in the control group reached 35.00±0.70%, while the 2 mg/L group dropped to 21.00±1.41%. Haemoglobin levels decreased from 13.50±0.70 g/dL in the control to 7.00±0.00 g/dL in the 2 mg/L group. The RBC counts reduced from 10.00±0.00 × 10^6/µL (control) to 4.00±0.00 × 10^6/µL (2 mg/L group). The WBC followed the same pattern with a decline from 13.50±0.70 × 10^3/µL to 5.50±0.70 × 10^3/µL. Other indices, such as Mean Corpuscular Volume, MCHC and lymphocyte percentages, varied significantly with Chromium exposure (Table 1).

At 28 days, the impacts of Chromium exposure became more severe. The PCV in the control group was 36.00±0.00%, dropping to 17.50% in the 2 mg/L group. Haemoglobin followed a similar pattern, with 15.00±1.41 g/dL (control) and 5.00±0.00 g/dL (2 mg/L group). The RBC counts decreased to 13.00±1.41 × 10^6/µL (control) and 4.00±0.00 × 10^6/µL (2 mg/L group). The WBC counts dropped to 4.50±0.70 × 10^3/µL in the 2 mg/L group, compared to 13.50±0.70 × 10^3/µL in the control. The MCH, lymphocyte, neutrophil, and monocyte count showed significant reductions with higher concentrations (Table 1).

Histopathological analysis

Muscle tissues showed myofiber disorganization, fibrosis, and necrosis. At lower concentrations, minimal disorganization and focal necrosis were present, while higher concentrations resulted in more severe disorganization and necrotic changes (Figure 1).

Histopathological analysis revealed consistent alterations in gill tissues, such as epithelial lifting, lamellar fusion, and hyperplasia. At 0.5 mg/L, minor changes were noted. However, at 1.0 mg/L and higher concentrations, hyperplasia and lamellar fusion became more severe, accompanied by increased epithelial proliferation (Figure 2).

Liver tissue exhibited hepatocellular vacuolation, sinusoidal dilation, and inflammatory infiltration. These changes were concentration dependent. At lower Chromium concentrations, vacuolation was more evident, whereas higher concentrations led to intensified hepatocellular necrosis and inflammation (Figure 3).

Growth performance indicators

Mean weight gain ranged from 9 g (in the 1.5 mg/L Cr group) to 15 g (control). Percentage weight gain followed a similar pattern, ranging from 68.44% to 113.21%. Specific growth rate ranged from 1.90% to 2.81%, with the highest SGR at 0.5 mg/L. The FCR remained consistent at 0.02 across all groups, indicating efficient feed utilization (Table 2).

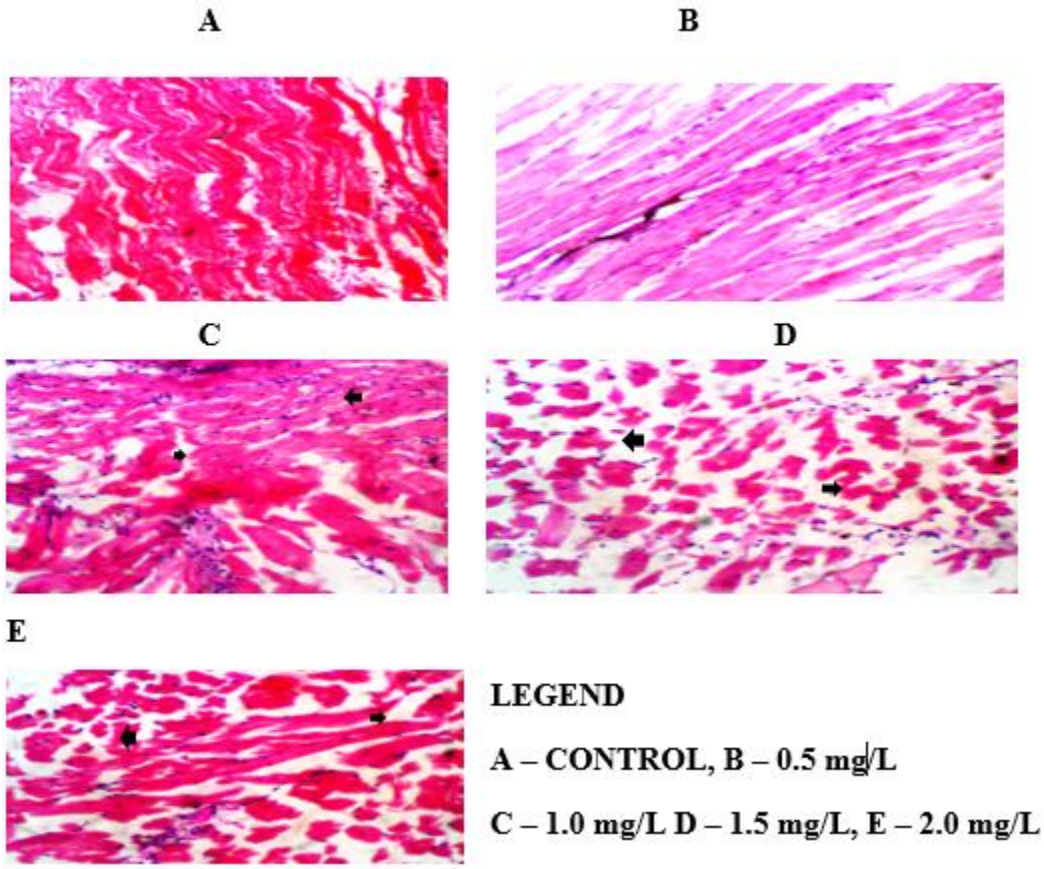

Figure 1: Photomicrograph of muscles of *Clarias gariepinus* juveniles (×400) exposed to sub lethal Chromium concentrations showing: Control (A): no observable lesion; 0.5mg/L (B): no observable lesion; 1.0mg/L (C): myofiber disorganization; 1.5mg/L (D): myofiber disorganization, disruption of muscle fibre; 2.0mg/L (E): focal necrosis within muscle tissues.

Table 1: Haematological parameters of *Clarias gariepinus* juveniles exposed to different concentrations of Chromium

Parameters	Control	0.5 mg/L	1 mg/L	1.5 mg/L	2 mg/L
			At 7 days		
PCV(%)	33.00±1.41^{c}	29.00±1.41^{b}	29.00±1.41^{b}	26.50±2.12^{b}	22.50±0.70^{a}
HB (g/dL)	11.50±0.70^{c}	9.50±0.70^{b}	8.50±0.70^{ab}	7.50±0.70^{a}	8.50±0.70^{ab}
RBC (10^{6}/µL)	9.50±0.70^{c}	7.00±0.00^{b}	6.50±2.12^{b}	5.50±0.70^{ab}	4.50±0.00^{a}
WBC (10^{3}/µL)	11.50±0.70^{a}	10.50±2.12^{a}	9.50±2.12^{a}	9.00±2.82^{a}	8.00±4.24^{a}
PLATELETS (10^{3}/µL)	9.50±0.70^{c}	7.50±0.70^{bc}	6.50±2.12^{ab}	5.00±0.00^{ab}	4.00±0.00^{a}
MCH (pg)	46.50±2.12^{d}	43.50±0.70^{d}	39.50±0.70^{c}	35.50±0.70^{b}	31.00±1.41^{a}
MCV (fL)	14.50±0.70^{e}	12.00±0.00^{d}	9.50±0.70^{c}	6.50±0.70^{b}	5.00±0.00^{a}
MCHC (g/dL)	39.50±0.70^{c}	34.00±1.41^{b}	32.00±0.00^{b}	27.50±0.70^{a}	25.50±0.70^{a}
LYMP (%)	44.00±1.41^{e}	41.50±0.70^{d}	39.00±0.00^{c}	35.50±0.70^{b}	33.00±0.00^{a}
NEU (%)	60.50±0.70^{d}	57.00±1.41^{c}	55.50±0.70^{c}	49.50±0.70^{b}	44.50±2.12^{a}
MON (%)	2.00±0.00^{c}	1.50±0.70^{bc}	1.50±0.70^{bc}	0.50±0.70^{ab}	0.00±0.00^{a}
			At 14 days		
PCV(%)	35.00±1.41^{d}	31.00±1.41^{c}	27.5±0.70^{b}	24.00±1.41^{a}	21.00±1.41^{a}
HB (g/dL)	13.50±0.70^{c}	9.50±0.70^{b}	9.00±0.00^{b}	9.56±0.62^{b}	7.00±0.00^{a}
RBC (10^{6}/µL)	10.00±0.00^{c}	6.00±0.00^{b}	5.50±0.70^{b}	6.01±0.29^{b}	4.00±0.00^{a}
WBC (10^{3}/µL)	13.50±0.70^{c}	9.50±2.12^{b}	9.50±0.70^{b}	8.50±0.70^{b}	5.50±0.70^{a}
PLATELETS (10^{3}/µL)	10.50±0.70^{c}	7.00±0.35^{b}	7.55±0.35^{b}	4.50±0.70^{a}	4.00±0.00^{a}
MCH (pg)	45.50±3.53^{c}	39.50±0.70^{b}	37.50±0.70^{bc}	32.50±3.53^{ab}	29.50±0.70^{a}
MCV (fL)	14.50±2.12^{c}	10.50±0.70^{bc}	9.50±0.70^{bc}	8.00±4.24^{ab}	4.50±0.70^{a}
MCHC (g/dL)	44.50±0.70^{c}	31.50±2.12^{b}	30.50±0.70^{b}	28.00±4.24^{ab}	24.50±0.70^{a}
LYMP (%)	44.00±1.41^{c}	40.00±0.00^{bc}	39.00±1.41^{bc}	37.00±4.24^{b}	31.00±1.41^{a}
NEU (%)	60.50±0.70^{c}	53.00±0.00^{b}	52.50±0.70^{b}	52.00±2.82^{b}	39.50±0.70^{a}
MON (%)	2.00±0.00^{b}	1.00±0.00^{a}	1.00±0.00^{a}	0.00±0.00^{c}	0.00±0.70^{c}
			At 28 days		
PCV(%)	36.00±0.00^{d}	27.50±3.53^{c}	25.00±1.41^{bc}	21.50±0.70^{ab}	17.50±0.70^{a}
HB (g/dL)	15.00±1.41^{b}	8.00±1.41^{a}	7.00±2.82^{a}	6.00±1.41^{a}	5.00±0.00^{a}
RBC (10^{6}/µL)	13.00±1.41^{c}	6.00±0.00^{b}	5.00±0.00^{ab}	4.50±0.70^{ab}	4.00±0.00^{a}
WBC (10^{3}/µL)	13.50±0.70^{c}	8.00±1.41^{b}	7.00±1.41^{ab}	5.50±0.70^{ab}	4.50±0.70^{a}
PLATELETS (10^{3}/µL)	10.50±0.70^{d}	7.00±0.00^{c}	5.00±0.00^{b}	3.50±0.70^{a}	4.00±0.00^{ab}
MCH (pg)	46.00±2.12^{d}	37.50±0.70^{c}	34.50±2.12^{bc}	31.00±1.41^{ab}	29.50±0.70^{a}
MCV (fL)	14.50±0.70^{c}	10.50±0.70^{b}	9.00±0.00^{b}	6.50±0.70^{a}	5.50±0.70^{a}
MCHC (g/dL)	35.00±1.41^{b}	29.50±2.12^{b}	26.50±0.70^{b}	23.00±0.00^{a}	20.50±0.70^{a}
LYMP (%)	45.00±1.4^{d}	37.50±0.70^{c}	33.50±2.12^{b}	32.00±1.41^{ab}	29.00±1.41^{a}
NEU (%)	59.00±1.41^{d}	50.00±0.00^{c}	48.50±0.70^{c}	45.00±1.41^{b}	41.00±1.41^{a}
MON (%)	2.00±0.00^{b}	1.00±0.00^{ab}	0.50±0.70^{a}	0.50±0.70^{a}	0.00±0.00^{a}

Mean value in each row with similar superscripts are not significantly different

PCV = Packed Cell Volume; HB = Haemoglobin; RBC = Red Blood Cell; WBC = White Blood Cell; MCV = Mean Corpuscular Volume; MCH = Mean Corpuscular Haemoglobin; MCHC = Mean Corpuscular Haemoglobin Concentration; NEU = Neutrophils; LYMP = Lymphocytes; MONO = Monocytes.

Table 2: Growth Vriables of *Clarias gariepinus* exposed to Sublethal Concentrations of Chromium

Treatment	Initial weight(g)	Final weight (g)	Weight gain (%)	Mean weight gain (g)	Specific growth rate	Feed conversion ratio
Control	13.25±0.23^{b}	28.25±0.50^{e}	113.21±2.50^{e}	15.00±0.80^{e}	1.90±0.03^{a}	0.02±0.01^{a}
0.5mg/L	12.40±0.11^{a}	23.90±1.00^{b}	92.74.60±3.60^{d}	11.50±0.30^{d}	2.81±0.08^{a}	0.02±0.00^{a}
1mg/L	13.25±0.14^{b}	23.25±1.50^{c}	75.47±5.50^{c}	10.00±0.53^{c}	2.41±0.05^{a}	0.03±0.00^{a}
1.5mg/L	13.15±0.00^{b}	22.15±0.80^{d}	68.44±4.00^{a}	9.00±0.87^{b}	2.27±0.05^{ab}	0.03±0.00^{a}
2.0mg/L	12.40±1.00^{a}	21.40±0.70^{a}	72.58±2.80^{b}	9.00±0.20^{a}	1.99±0.02^{a}	0.03±0.00^{a}

Mean value in each column with similar superscripts are not significantly different

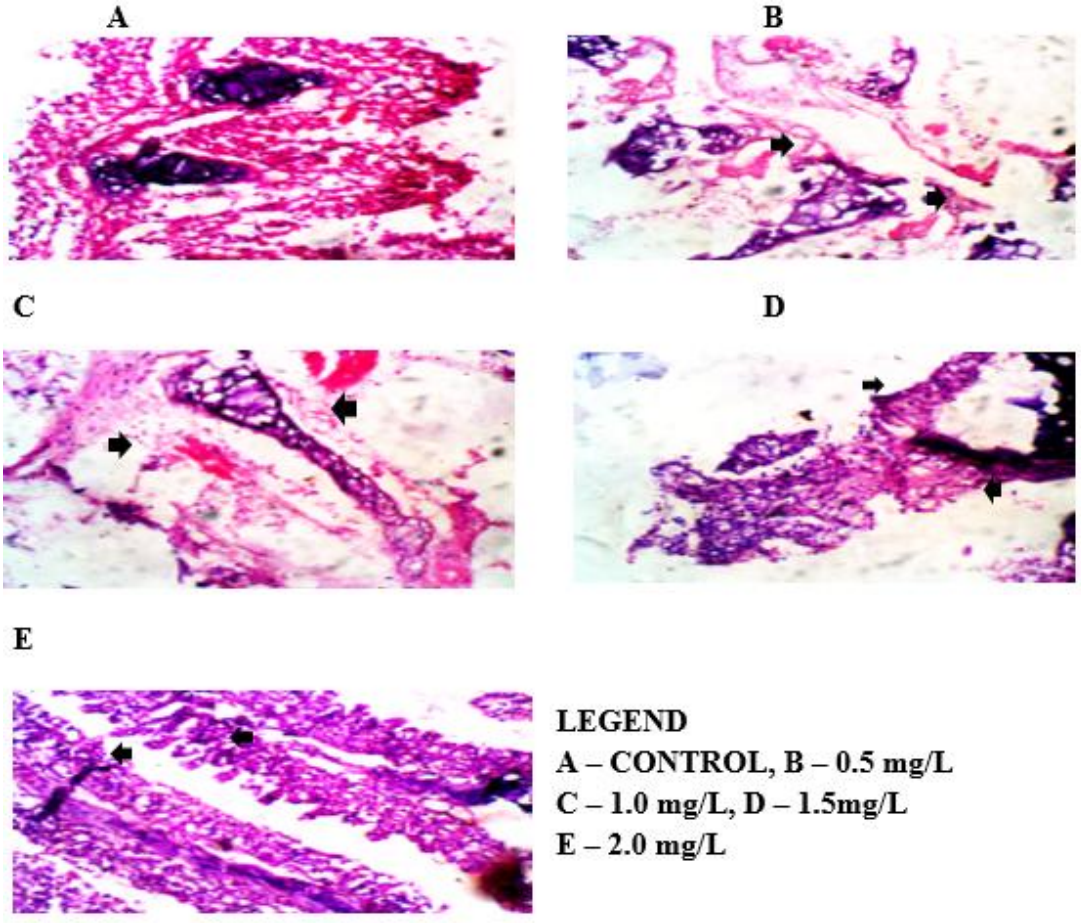

Figure 2: Photomicrograph of gills of *Clarias gariepinus* juveniles (×400) exposed to sub lethal Chromium concentrations showing; Control (A): no observable lesion; 0.5mg/L (B): mild lamellar fusion; 1.0mg/L (C): epithelial lifting and lamellar fusion; 1.5mg/L (D): extensive epithelial lifting; 2.0mg/L (E): hyperplasia

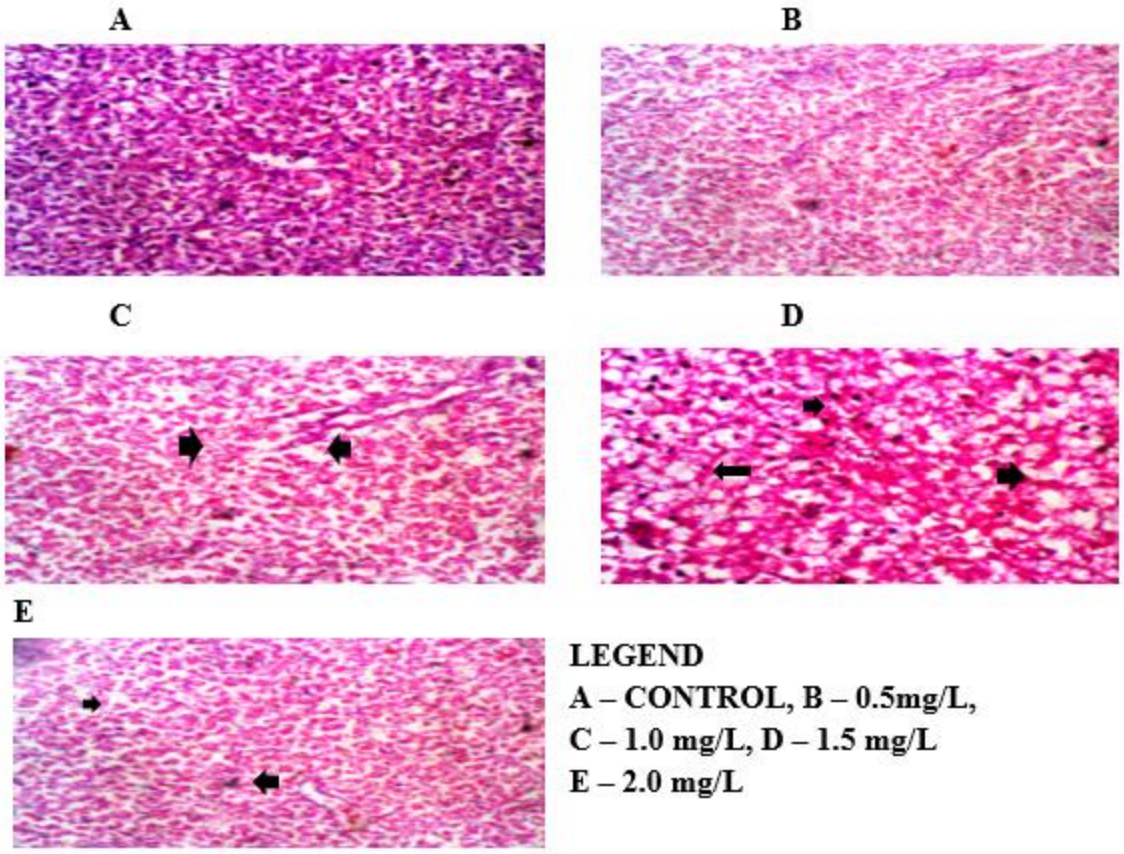

Figure 3: Photomicrograph of liver of *Clarias gariepinus* juveniles (×400) exposed to sub lethal Chromium concentrations showing - Control (A): no observable lesion; 0.5mg/L (B): no observable lesion; 1.0mg/L (C): mild hepatocellular vacuolation; 1.5mg/L (D): excessive hepatocellular vacuolation; 2.0mg/L (E): atrophy of hepatocytes.

DISCUSSION

The parameters measured are critical indicators of fish health, reflecting physiological responses to environmental stressors such as heavy metal contamination. The haematological parameters demonstrated dose- and time-dependent alterations in response to Chromium, indicating disturbances, histopathological damage and growth inhibition. Increasing Chromium concentrations caused significant haematological disruptions, particularly in PCV, HB, RBC, WBC, and platelet count. This dose-dependent decline suggests hematotoxic effects of Chromium, leading to anaemia and immune suppression in *Clarias* juveniles (Islam *et al*., 2020; Witeska *et al*., 2023). Reduced PCV and HB levels suggest impaired oxygen transport capacity, potentially affecting metabolic processes (Adeyemo, 2005; Hashim *et al.,* 2022). Similarly, *Cyprinus carpio* exposed to heavy metals showed marked reductions in RBC, HB, MCV, and PCV, indicating of a broad-spectrum anaemia (Vinodhini and Narayanan, 2008). The decreased RBC and platelet count corroborate previous findings on fish exposed to cadmium and other metals,

which also reported erythrocyte damage, accelerated destruction, and impaired erythropoiesis (Kori-Siakpere *et al.,* 2009; Witeska *et al.,* 2023).

In comparison to recent studies, the observed reductions in haematological indices were more pronounced, particularly the 74.15% decrease in WBC at higher concentrations. This deviation may be linked to species-specific sensitivities, differences in experimental designs, or environmental factors like water quality (Ahmed *et al*., 2022). *Clarias* juveniles, known for their tolerance to polluted environments, may experience heightened susceptibility under chronic Chromium exposure, due to potential disruptions in metal detoxification pathways (Al-Asgah *et al.,* 2015). Studies by Nafees *et al.* (2023) and Aziz *et al.* (2023) on heavy metal exposure in *Oreochromis niloticus* showed lower reductions in WBCs compared to the findings of this study, possibly due to variations in the immune resilience of different species or the presence of other stressors in their environments.

Moreover, the altered MCH, MCV, and MCHC values reflect aberrant erythropoiesis and haemoglobin synthesis, indicating that Chromium impairs both the production and function of erythrocytes. This aligns with earlier studies in *Tilapia sparmanii* and *Cyprinus carpio* (Wepener, 1990; Martin and Krol, 2017), where toxicants interfered with iron metabolism and erythrocyte membrane stability. These led to abnormal erythrocyte size and haemoglobin content. These haematological disruptions are not only indicative of anaemia but also suggest potential Chromium-induced oxidative stress on haematopoietic tissues (Crafford and Avenant-Oldewage, 2010).

This study also aligns with the immune suppression reported in previous studies. The decreased WBC counts, particularly in LYMP and NEU, suggest that Chromium compromises the immune system by disrupting leukocyte production and function (Segner *et al.,* 2011). This suppression may be due to oxidative damage to hematopoietic tissues like the spleen and kidney (Witeska *et al.,* 2023). This immunosuppression could impair the fish's ability to mount an effective immune response, increasing susceptibility to infections in contaminated environments.

The gills, being the primary site of metal accumulation, exhibited epithelial lifting, lamellar fusion, and hyperplasia, with severity increasing with concentration. These pathologies, indicative of compromised respiratory and osmoregulatory function, are consistent with the chronic effects of heavy metal exposure (Shahid *et al.,* 2021). Similar histological damage was observed in *O. niloticus* exposed to chlorpyrifos and lead, where significant changes in gill structure, including necrosis and mucus cell proliferation, were reported (Paul *et al.,* 2019; Hossain *et al.,* 2022).

Notably, epithelial lifting and lamellar fusion were more pronounced in this study compared to similar reports on *Cyprinus carpio* exposed to Lead (Barbieri, 2016; Kamila *et al*., 2023). This deviation may be due to the differing physiological adaptations between species, with *Clarias* potentially being more susceptible to Chromium-induced ion regulatory dysfunction, given its

preference for low-oxygen environments. Additionally, Cr may have a stronger affinity for binding to epithelial tissues in *Clarias* juveniles, resulting in more extensive gill damage compared to species like *Cyprinus* or *Tilapia*.

The liver, a key detoxification organ, showed marked vacuolation, sinusoidal dilation, and inflammatory cell infiltration, indicating progressive liver damage due to Chromium exposure. These changes are consistent with those observed in *O. niloticus* exposed to Pb, where vacuolation and necrosis were prominent (Kiran *et al.,* 2021). The degree of necrosis was more severe at higher Chromium concentration (2 mg/L), suggesting a dose-dependent exacerbation of liver pathology.

This study supports the hypothesis that Chromium induces oxidative stress and disrupts hepatic metabolism, leading to inflammatory responses and cellular damage (Cullen and Stalker, 2016). The severity of these histopathological changes is comparable to those reported in fish exposed to similar heavy metals, such as Copper and Cadmium (Younis *et al.,* 2013). However, the more severe vacuolation and sinusoidal dilation observed in *Clarias* juveniles may be attributed to the species' high metabolic demand and detoxification capacity, which renders it more susceptible to liver damage under chronic Cr exposure.

Although less pronounced, muscle tissue changes, including myofiber disorganization and necrosis, were observed, particularly at higher Chromium concentrations. These findings suggest that Cr toxicity extends to skeletal muscles, impairing swimming performance and overall fitness (Goody *et al.,* 2017). Shah *et al.*, (2021), reported similar muscle fibre degeneration in *Labeo rohita* exposed to Chromium. The muscle damage in this study was more severe, potentially due to longer exposure durations or higher Chromium bioaccumulation in *Clarias* muscles.

The growth performance of *Clarias* juveniles was adversely affected by Chromium exposure, with reductions in final weight, percentage weight gain, and specific growth rate (SGR), particularly at higher concentrations. Ezejiofor *et al.* (2022) and Aziz *et al.* (2023), reported growth retardation in fish exposed to heavy metals. The observed decline in feed conversion ratio (FCR) suggests that Chromium disrupts nutrient absorption and metabolic efficiency, leading to poor growth performance. The decreased growth rates were more pronounced than those reported for other species like *O. niloticus* and *L. rohita*, likely due to the unique physiological demands of *Clarias* in polluted environments. The higher tolerance of *Clarias* to low oxygen and polluted waters may paradoxically render it more vulnerable to chronic Chromium toxicity, resulting in more pronounced growth impairments under prolonged exposure (Jan *et al.,* 2015; Kumar *et al.,* 2021).

CONCLUSION

The study demonstrated the detrimental effects of Chromium exposure on *Clarias gariepinus* juveniles, highlighting significant haematological, histopathological, and

growth impairments. Chromium exposure led to dose-dependent hematotoxicity, including anaemia-like conditions and immune suppression, as evidenced by alterations in PCV, HB, RBC, WBC, and other blood indices. Histopathological damage was observed in the gills, liver, and muscles, impairing organ function. The Chromium exposure significantly inhibited growth, with reductions in weight gain and specific growth rate, indicating compromised metabolic functions and overall health. These findings underscore the harmful impact of Chromium on fish in contaminated environments.

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